

# Armed Forces College of Medicine AFCM



# Case study Medical Microbiology & Immunology department



Male patient 32 years old with irrelevant past history presented to ER complaining of respiratory distress. The patient informs the physician that he suffered from rapid onset 6 days duration of parathesia then weakness in both upper limbs and lower limbs. Upon examination the ER physician found that the patient blood pressure is 90/50, temp. 37c, blood sugar 110. The physician ask about any symptoms of upper respiratory tract infection, astrophtoritis or vaccination in the last 3 weeks

#### Case



The motor examination shows hypotonia, hyporeflexia, proximal and distal weakness. There is no atrophy, hypertrophy fasciculations. The patient has positive radicular stretch. He loses both superficial and deep sensation (radicular distribution). The provisional diagnosis is GBS.

#### The plan of management is:

- Admission to ICU.
- Full lab.
- Lumber puncture.



#### ?What are the gradings of weakness

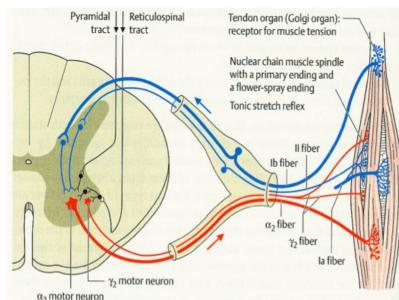
#### Scoring muscle strength

- > 0 = No movement
- >1 = Flicker movement
- ≥2 = Movement when gravity is eliminated
- → 3 = Movement against gravity but not against examiner resistance
- ►4 = Movement against resistance but weaker than normal
- ➤ 5 = Full movement against full resistance



# What are the interpretation of reduced tone?

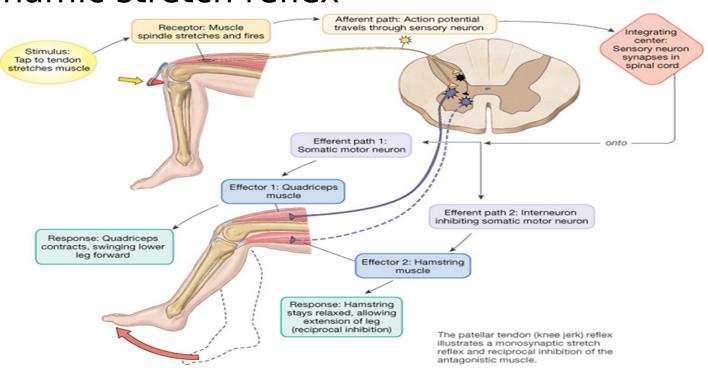
- The muscle tone is a static stretch reflex
- Stimulus: maintained muscle stretch
- Receptors: nuclear chain intrafusal fibers
- Afferent: type II fibers
- Center: alpha motor neuron
- Efferent: A alpha motor neurons
- Response: tonic mild muscle contraction
- Any disease that affect Lower motor neuron (the efferent) leads to reduction in the muscle tone





.Knee and ankle reflex jerks are absent

What are the interpretation of absent jerks?
Dynamic stretch reflex

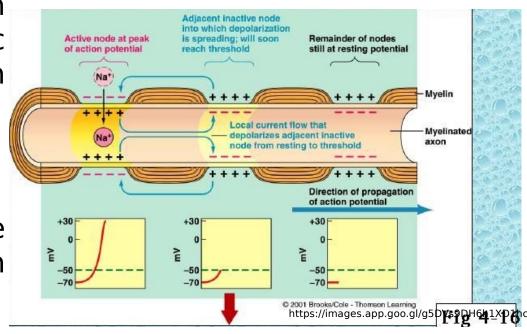




# What is the pathophysiological changed that resulted in this condition?

 Presence of myelin sheath allows fast and economic nerve impulses conduction by saltatory conduction

 Myelin sheath damage affect nerve conduction velocity





# Is it upper or lower neuron lesion? Justify your answer



- This is a lower motor neuron case.
- Localized upper and lower limb weakness.
- Presence of hypotonia and hyporeflexia.



Patient feet is painful although there is impaired pinprick sensation up to the thighs and reduced joint position sense and vibration sense in the ankles.

**Explain** 



- Pinprick is a fast pain sensation carried by A delta fibers which are myelinated nerves
- Also vibration sense is carried by A beta thick myelinated nerves
- The inflammatory condition will be associated with slow pain, that is carried through slow c un-myelinated fibers



Interpret the symptoms and signs of patient based on the myelin function



- This is a Demyelinating disease leading to
- Sensory deficits: lesion in dorsal root myelinated fibers (A delta, A beta) leads to loss of fast pain sensation, vibration sense
- Motor deficits: lesion in A alpha fibers
- voluntary movement: weakness in muscle power
- Stretch reflex: absent; hypotonia, hyporeflexia



# what is the clinical presentation of ?GBS



- Rapid onset,
- Regressive course,
- Ascending quadriplegia,
- Loss of superficial and deep sensation,
- Cranial nerve affection (3<sup>rd</sup>, 6<sup>th</sup>, 7<sup>th</sup>, 9<sup>th</sup> and 10<sup>th</sup>),
- Radicular pain.



# ?What is the work up for diagnosis of GBS



Detailed history and clinical examination, SCF sampling and analysis, nerve conduction study and electromyography.



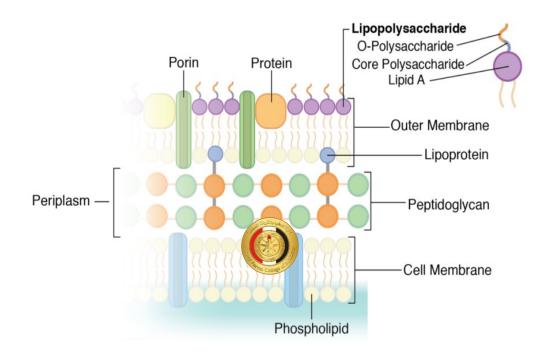
# What is the most probable causative organism of gastroenteritis?



The most probable causative organism of this gastroenteritis is Campylobacter jejuni (C. jejuni).

- Gram-negative rods.
- Comma- or S shaped.
- Microaerophilic, growing best in 5% oxygen rather than in the 20% present in the atmosphere.
- Optimum temp: 42°C.



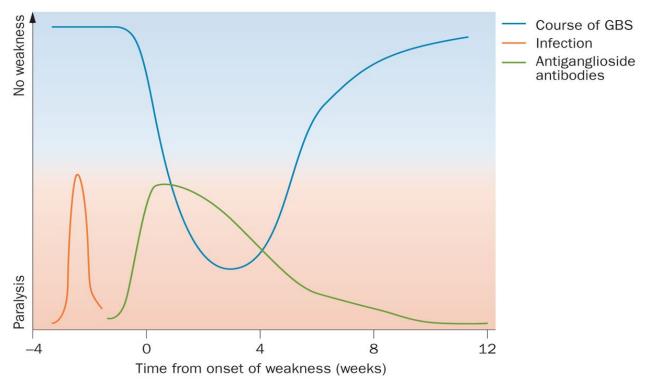


Gram Negative Bacteria Cell Wall



What is the pathogenesis of GBS in relation to this organism?





The majority of patients with GBS report an infection before the onset of weakness. Antiganglioside antibodies are often detected; their levels decrease over time. Progressive weakness reaches its maximum within 4 weeks. The recovery phase may last many weeks, months or even years

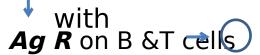
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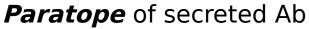
# **Epitope (antigenic determinant)**

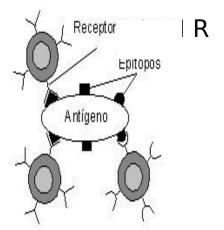


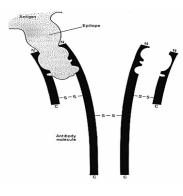
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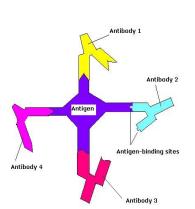
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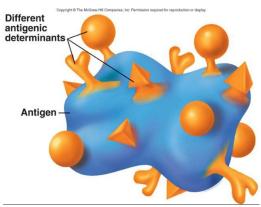


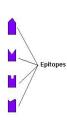












## Heterophil Antigens (cross reactivity)

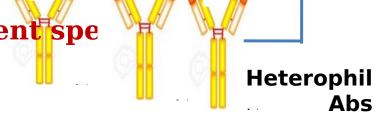
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#### **I-Characters**

**A- Derived from different spe**Organisms, mammals



3

#### **B-Share one or more epitopes**

Induce production of heterophil Abs

Cross react with shared epitopes

#### **II-Practical Applications**

#### Pathogenesis of some autoimmune diseases



Some C. jejuni lipo-oligosaccharides

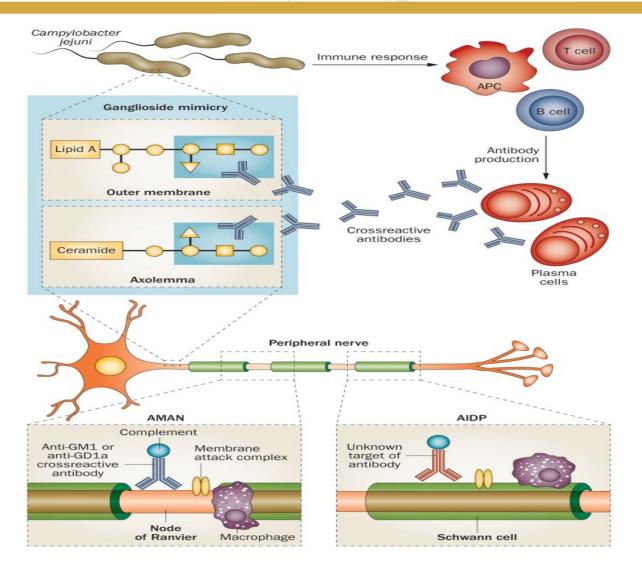


activate dendritic cells via Toll-like receptor 4 & CD14

type 1 interferen & tumour necrosis factor (TNF)

#### antibodies





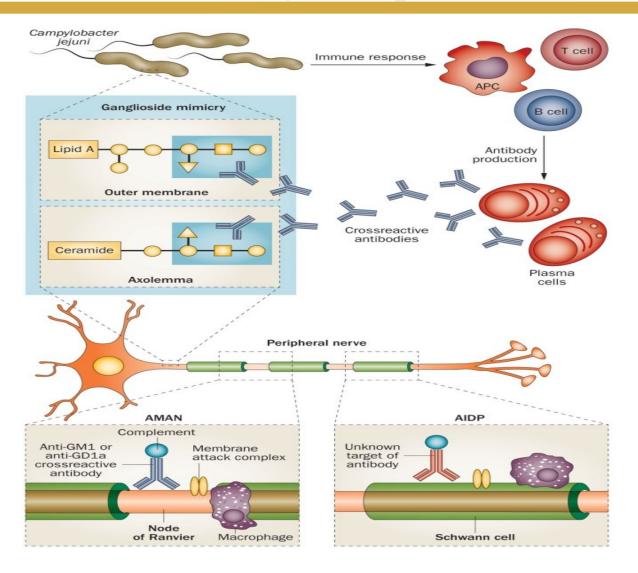
**Immunopathogenesis of GBS** 



# **Molecular mimicry:**

Infections with Cipinal production of antibodies (anti-GM1 and anti-GD1a antibodies) crossreact with gangliosides (GM1 and GD1a on peripheratives) Complement activation detachment of paranodal myelin and nerve conduction failure. Macrophages then invade from the nodes scavenging the injured axons vesicular degeneration.





**Immunopathogenesis of GBS** 



What is the possible relation between respiratory tract infection & GBS?



GBS can occur after infection with other pathogens that can cause respiratory tract infections as:

- Cytomegalovirus.
- Epstein- Barr virus.
- Mycoplasma pneumonia.
- Haemophilus influenzae.
- Influenza A virus.
- Some researches reported that GBS can occur post COVID 19 infection.